Getting to Basics

HEALTH CARE for the poor, the young, the aging often means illness care. Even the very best care is not enough to help these most vulnerable, for they may be irretrievably sick before they receive attention. Illness care is necessary when safety nets, systems, and bodies break. We are faced with disturbing questions about this breakage.

- Why are "at least 4 million Americans (and possibly as many as 14 million) . . . now living on the knife edge of homelessness, already doubled-up with friends and family or one paycheck away from not being able to pay their rent?"[1]
- Even though $3 in short-term hospital costs are saved for every $1 spent on the prenatal portion of the Women, Infants, and Children Supplemental Food Program, why did only 59% of potentially eligible women and children receive this help in 1989?[2]
- Why do over 20 million Americans need drug and alcohol treatment?[3] Why is so little care available, especially for women, adolescents, and minorities?
- Why do African-American men in Harlem have a shorter life expectancy after age 40 than men in Bangladesh, with cardiovascular disease and cirrhosis the main causes of excess mortality?[4]
- Why are infant death rates in some parts of the United States higher than those in many Third World countries?
- Why is the number of Americans without any health care coverage increasing? Why is so little research undertaken in diseases of aging?

How did we get into the situation in which so much suffering occurs before health or illness care is received? Reasons seem plentiful. Federal spending for education training, employment, and human services has plummeted over $82.3 billion since 1982.[5] Military spending has grown. Bailouts and war have been undertaken. We are faced with conflicting, bewildering demands. We wallow in indecision. Private giving has not kept up with the need, nor can it. Brian O'Connell, president of Independent Sector, a national coalition of foundations, philanthropic corporations, and not-for-profit organizations, has noted that the voluntary sector has enormous vitality and provides crucial services but has limits. For example, nonprofit groups are able to spend about $250 billion per year compared to governmental spending of $2.5 trillion.[6]

Our national assets are plentiful. We have talent, money, natural resources, creativity. We can solve problems. We know what is right. We know that good food, homes, usable skills, and self-esteem will help the poor, the young, the aging. We know that failing to invest in these has led to unnecessary suffering for millions. Good health comes more easily to people who are well-nourished and well-clothed, to people who respect themselves. The basic issue is not health or illness care. The basic issue precedes the point at which this care must intervene. The basic issue is caring enough about the vulnerable to give them a hand. We do care, of course. We recognize the consequences of not investing in people. We must now pull together, clarify our values, reassert that vulnerable people are a high priority, and move ahead.

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Meningeal Carcinomatosis

MENINGEAL CARCINOMATOSIS occurs when carcinomas, gliomas, sarcomas, melanomas, or lymphomas diffusely infiltrate the leptomeninges and subarachnoid space to produce a syndrome of chronic meningitis which may simulate that caused by fungi, tuberculosis, sarcoidosis, and meningovascular syphilis.[1,2] Although meningeal carcinomatosis was first reported with gastric carcinoma, today the more common tumors are usually responsible. In their series of 90 patients with meningeal carcinomatosis, Wasserstrom and co-workers reported that 46 patients had breast, 23 had lung, and 11 had melanoma primaries.[1] Evidence suggests that the incidence of meningeal carcinomatosis increases with longer survival.[3,4] Indeed, meningeal carcinomatosis primarily occurs late in the course of most patients' disease.

The pathogenesis of meningeal carcinomatosis is imperfectly understood. Recent data suggest that tumor enters the leptomeninges through several pathways, in part depending on the primary tumor type.[5] Lung and breast tumors most commonly invade from vertebral and paravertebral metastases; gastrointestinal cancer enters through perineural spaces. When deep central nervous system (CNS) parenchymal metastases are present, meningeal carcinomatosis follows cancer metastasis by the arterial route. The pathophysiology of clinical signs depends on local infiltration of the cortex, spinal cord, and cranial and spinal nerves. The usual regions of dense tumor growth are along the base of the brain and the dorsum of the spinal cord. Tumor growth along the base may obstruct cerebrospinal fluid (CSF) pathways leading to raised intracranial pressure and hydrocephalus. Experimental studies have elucidated some of the mechanisms responsible for the pathophysiology of the disease. In a model of B16 mouse melanoma-induced meningeal carcinomatosis, there was alteration of the blood-brain-CSF barrier, which allowed entry of the water-soluble drug, doxorubicin (Adriamycin).[6] In a rat model of meningeal carcinomatosis, the disease selectively lowered glucose use both in brain regions underlying the tumor and remote regions anatomically related to the former, helping to explain the diversity of neurologic dysfunction seen in patients.[7]

The clinical characteristics follow the pathogenesis; there is involvement of more than one CNS location—cerebral, cranial nerves, spinal cord, and spinal nerves. Common manifestations include headache, mental changes, cranial nerve palsies, weakness and areflexia, and minimal signs of meningeal irritation. Areflexia is common, as are urinary retention and other autonomic signs. The CSF findings generally establish the diagnosis. Pressure may be normal or elevated, the glucose content is often below 2.5 mmol per liter (45 mg per dl), the protein content is usually elevated, and cell counts may reveal an increased number of mononuclear cells or cytologic evidence of malignant cells, or both. The last occurs at some time in 90% of patients.[1] Cultures are negative. Carcinoembryonic antigen (CEA) and β-glucuronidase may be found in the CSF of patients harboring CNS